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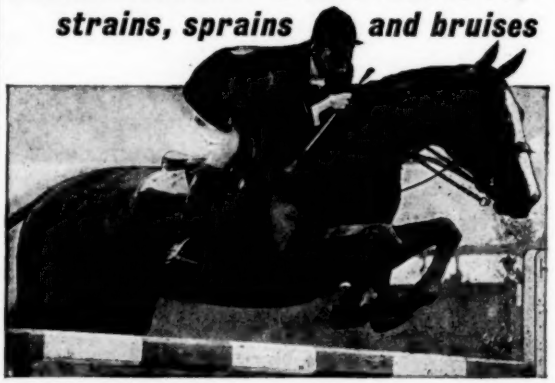

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Studies on Lungworm Infection of Pigs. II.—Lesions in Experimental Infections

BY

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Introduction

THE symptoms of lungworm (*Metastrongylus* spp.) infection of pigs and the long-standing lesions found in the lungs have been noted in studies of the life cycle of the parasites (Hobmaier & Hobmaier, 1929; Schwartz & Alicata, 1934; and Matoff, 1936) and in descriptions of natural infections (Lewis, 1926; Robertson, 1937; Schwartz, 1952; Dunn, Gentles & White, 1955,). Dunn (1956) has described the microscopical changes occurring in the late stages of the disease in pigs experimentally infected with lungworms. These included pulmonary lymphoid nodules, bronchitis, chronic emphysema, and bronchiolar muscular hypertrophy which are the changes associated with long-standing natural infections (Nieberle & Cohrs, 1931). Dunn also described the histological changes in the lungs of a pig killed 6 weeks after experimental infection which were significantly different from those in the long-standing cases. The gross and microscopical changes found in experimental infections of guinea-pigs have been described (Dunn & White, 1957) and these included pulmonary emphysema and consolidating bronchopneumonia in the developmental stages of the disease. A detailed examination of the progressive histopathology of all stages of the disease in the pig, similar to that carried out on *Dictyocaulus viviparus* infection of calves by Jarrett, McIntyre, and Urquhart (1957), has not been recorded.

The work described in this paper was carried out to study in more detail the character and distribution of the lesions occurring in the early patent phase of the disease in pigs experimentally infected with *Metastrongylus elongatus*.

Methods

Experimental Animals

The pigs used were from the Compton herd which is free from virus pneumonia. They were reared indoors on concrete on an artificial milk substitute diet after early weaning at two days of age. Details of these animals are given in Table I. All were clinically healthy when infected and examination of their faeces for Ascaris and Metastrongyle eggs was negative. The infective doses were administered orally through a drenching gag with the pig held upright and the head vertical. Water was given after the dose to ensure that all larvae were swallowed. All pigs were inspected daily and were killed for examination 35 days after infection.

Infective Larvae

Infective *M. elongatus* larvae were obtained from cultures of earthworms (*Eisenia foetida*) exposed to infection for 64 to 116 days. The larvae were liberated from the earthworms by cutting up the infected anterior parts of the worms in normal saline and allowing the mixture to stand at room temperature for several hours. The fragments were then digested in a pepsin-hydrochloric acid mixture at 37° C. so that the remaining larvae could be recovered. All infective larvae were pooled, washed in normal saline, and counted by a dilution method. Accurate scaled doses of viable infective larvae were then prepared, each dose contained in 20 ml. of normal saline. The numbers of larvae used are listed in Table I.

Laboratory Examinations

Parasitology. Examination of faeces samples to determine the patency of the infections was carried out by the flotation method of Dunn and White (1954) using a saturated magnesium sulphate solution. At *post-mortem* examination an estimate of the numbers of lungworms in the lobes of the left lung and in the intermediate lobe of the right lung was made by cutting open the bronchi with fine scissors, removing as many whole worms as possible, and counting them in isotonic saline in a Petri dish.

TABLE I

PARTICULARS OF PIGS EXPERIMENTALLY INFECTED WITH *M. Elongatus* LARVAE AND KILLED FOR EXAMINATION 35 DAYS AFTER INFECTION

Pig number	Age in weeks	Dose of infective larvae	Adult lungworms counted					Emphysema	Consolidation	Increase in size of bronchial lymph nodes
			L.A.	L.C.	L.D.	I	Total			
1	8	O (Control)	0	0	0	0	0	—	—	None
2	15	" "	0	0	0	0	0	—	—	"
3	8	200	0	0	10	0	10	+	—	"
4	15	250	1	0	15	0	16	+	—	Slight
5	15	500	10	3	40	0	53	++	+	"
6	15	1,000	23	0	118	12	153	+++	—	"
7	8	2,000	24	11	147	25	207	++++	+++	Doubled
8	8	4,000	15	31	168	22	236	++++	++	"
9	8	8,000	52	39	397	32	520	+++++	++++	Trebled

L.A., L.C., L.D., I = Left apical, cardiac and diaphragmatic lobes and intermediate lobe.
— = Absent.
+ to +++++ = Degree of change.

Bacteriology. Consolidated areas of lung were cultured aerobically on bacto-tryptose ox-blood agar at 37° C.

Histology. Blocks of tissue for histological examination were fixed in 10 per cent. formol saline, cleared in benzene, and embedded in paraffin wax under reduced pressure. Routine staining was by haematoxylin and orange G-eosin: special methods included periodic acid-Schiff, picro-Mallory, and eosin-phloxine-tartrazine. Eleven blocks were taken from the same positions in the right lung in each case. Bronchial lymph nodes, heart, liver, kidney, spleen, small and large intestines, and mesenteric lymph nodes were also examined.

Results

Clinical Examination

Only pig No. 9, Table I, which received 8,000 larvae, showed distinct signs of clinical disease within the 35-day experimental period. Coughing was noted from the 13th day onwards and became more pronounced as the disease progressed. The cough was spasmodic, husky, and indistinguishable from that of virus pneumonia. Slight dyspnoea was present and this became more apparent after exercise. General signs of unthriftiness appeared before the end of the experiment. Pigs No. 7 and 8 which received 2,000 and 4,000 larvae respectively coughed occasionally after the 21st day when hustled round the pig pen but were otherwise healthy. None of the other pigs showed evidence of clinical abnormality.

Embryonated metastrongyle eggs were found in the faeces of the pigs receiving 1,000 or more larvae, 28 days after infection, but examination of the faeces of those given smaller infecting doses was negative.

Post-mortem *Examination*

After slaughter all the infected pigs were found to have active lungworm infections whereas the lungs of the 2 control animals were normal. Pulmonary emphysema was present in all affected lungs and in some cases there was marginal consolidation. There was a clear correlation between the extent of the lesions and the size of the infecting dose, as shown in Fig. 1.

The emphysematous areas had a distinctive yellowish white appearance and were turgid and sponge-like in texture and crepitant on pressure. These lesions were directly attributable to the presence of lungworms in the small bronchi and bronchioles of the region so that they occurred in a lobular pattern in which the affected segments were well demarcated and the delineation enhanced by the slight elevation of the emphysematous tissue over the adjacent non-expanded lung (Fig. 2). The lesions occurred along the margins of the lung or on its surface, the deeper lung tissue being unaffected. The consolidated areas (Fig. 3) were reddish pink, well demarcated, and slightly depressed below the surrounding tissue. The cut surface was smooth or finely granular and occasional minute greyish nodules were visible. Pressure produced oedema fluid from the lung tissue or muco-pus from the bronchi.

The distribution of the lung lesions is shown in Fig. 1. The caudal extremity of the diaphragmatic lobe was primarily and consistently affected. This

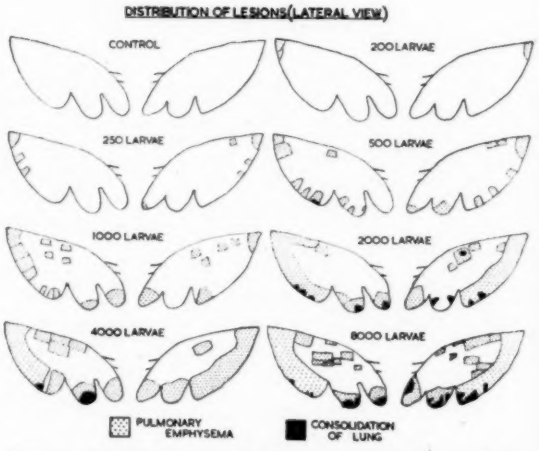

FIG. 1.—Distribution of the lesions in the lungs of pigs killed 35 days after oral administration of scaled doses of infective lungworm larvae.

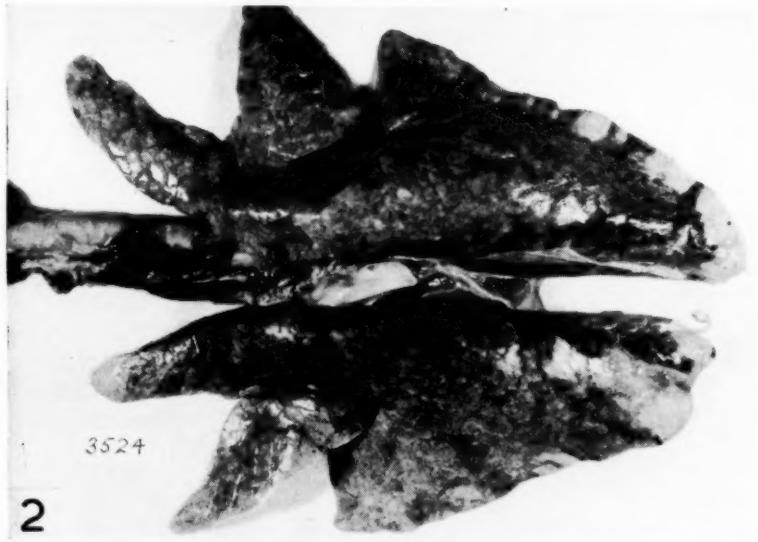

FIG. 2.—Lungs of pig 35 days after experimental lungworm infection with 250 larvae. There is lobular emphysema of the caudal extremities of the diaphragmatic lobes. 2/5ths actual size.

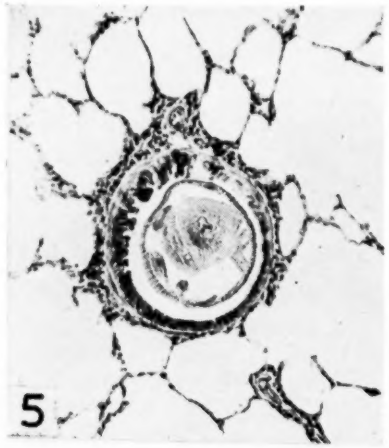

FIG. 5.—Cross-section of bronchiole containing anterior portion of lungworm. The surrounding lung is emphysematous. H. and E. × 100.

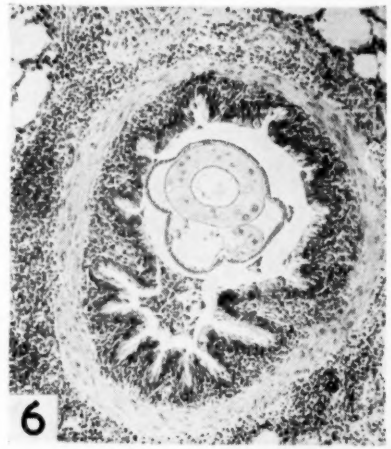

FIG. 6.—Section of large bronchiole containing lungworm and showing bronchiolar muscular hypertrophy. There is cellular infiltration of the mucosa and the surrounding connective tissue. H. and E. × 60.

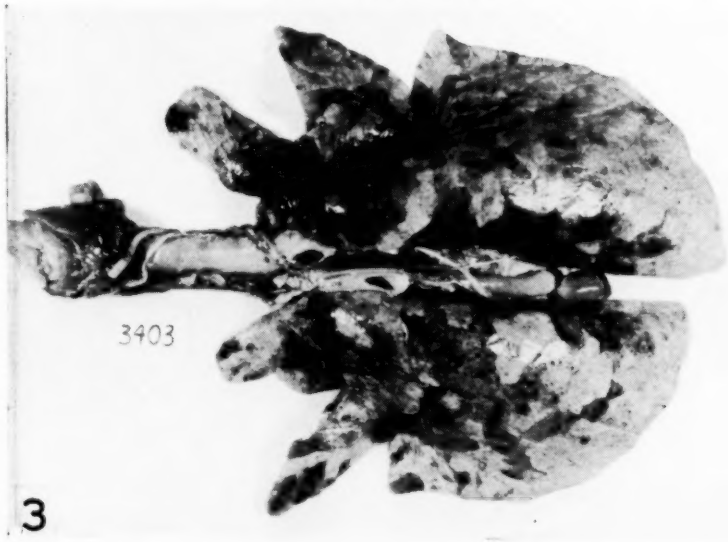

FIG. 3.—Lungs of pig 35 days after experimental lungworm infection with 8,000 larvae. Emphysema is present in all lobes and there is pulmonary consolidation in the ventral parts of the anterior lobes. 2/5ths actual size.

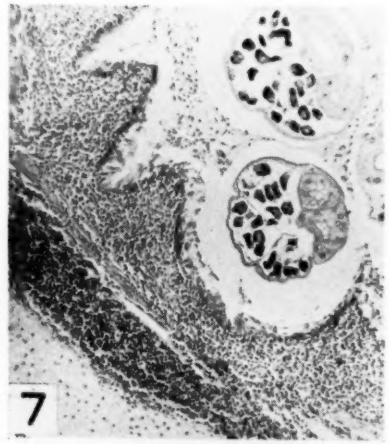

FIG. 7.—Wall of bronchus containing adult lungworms. There is mucoid change of the epithelium, cellular infiltration of the lamina propria, and early lymphoid hyperplasia in the adjacent connective tissue. H. and E. × 100.

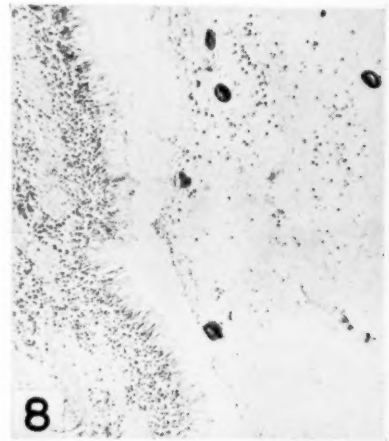

FIG. 8.—Embryonated lungworm eggs in mucous exudate in large bronchus. Mucoid change of the bronchial epithelium is pronounced. H. and E. × 60.

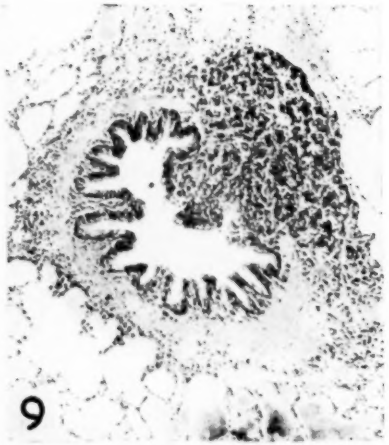

FIG. 9.—Early peribronchiolar lymphoid hyperplasia and bronchiolar smooth muscle hypertrophy. H. and E. × 60.

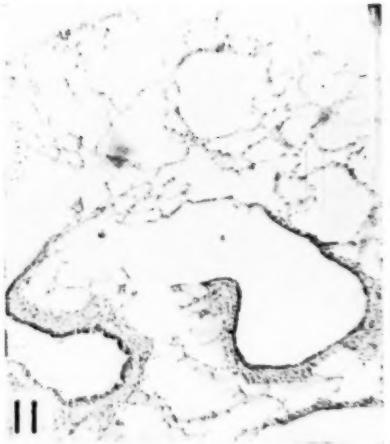

FIG. 11.—Pulmonary vesicular emphysema in lung lobule adjacent to obstructed bronchus. H. and E. × 50.

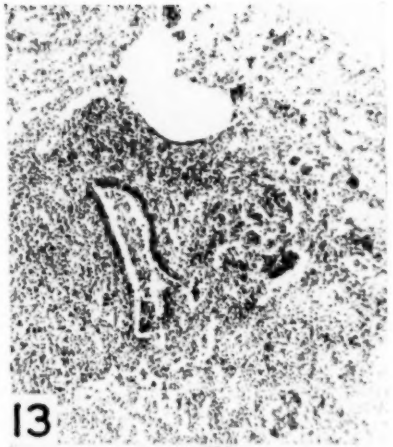

FIG. 13.—Area of pulmonary consolidation associated with reactive pneumonia in an anterior lobe of the lung. H. and E. × 60.

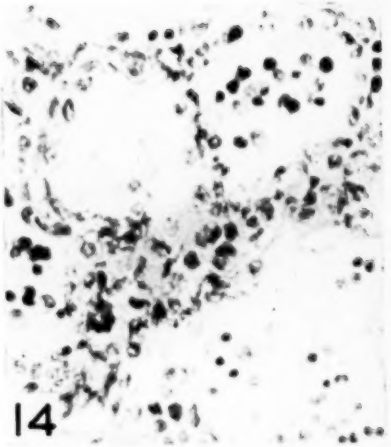

FIG. 14.—Cellular alveolar reaction in an area of pulmonary consolidation showing epithelialisation of alveoli. H. and E. × 350.

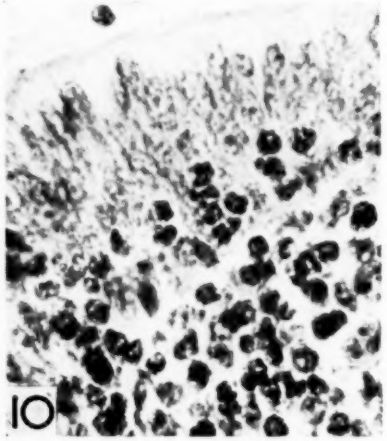

FIG. 10.—Cellular infiltration of the bronchial mucosa with eosinophil leucocytes and round cells. H. & E. × 450.

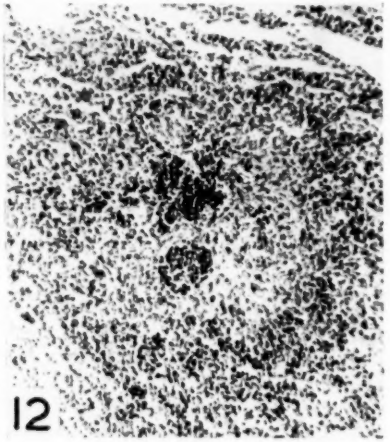

FIG. 12.—Eosinophil nodule in emphysematous part of the lung. The central masses of darkly staining cells are composed of eosinophil leucocytes. H. and E. × 100.

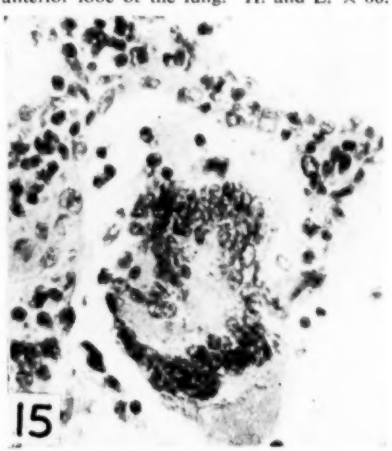

FIG. 15.—Large multinucleate giant-cell free in alveolus and cellular infiltration of the alveolar walls. H. and E. × 350.

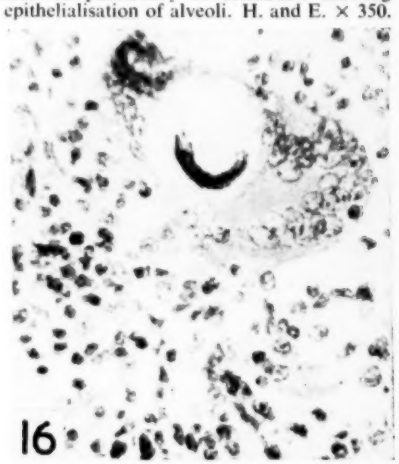

FIG. 16.—Phagocytosis of larva by alveolar giant-cell. The deeply staining cells in the field are eosinophil leucocytes. H. and E. × 350.

was followed by extension along the basal border and dorsal surface of the same lobe but only isolated lesions occurred in the anterior lobes in the light infections. The dissemination of lesions to all lobes became most complete with infective doses of 1,000 larvae and above. The diaphragmatic lobes were still most severely affected and the emphysematous areas along the basal border became confluent. The anterior lobes were affected in their ventral aspects. The distribution of the lung consolidation was different from that of the emphysema. Consolidation occurred in heavily infected lungs where the anterior lobes were involved and affected the ventral borders of the apical, cardiac, and intermediate lobes and the antero-ventral aspects of the diaphragmatic lobes. There was enlargement of the bronchial lymph nodes in the animals given the largest doses (see Table I) and the cut surfaces showed grey hyperplastic areas in the cortex. In addition several small "cysts" filled with clear fluid were found in the ileo-caecal lymph nodes of pigs Nos. 3, 7, 8, and 9. There were no apparent hepatic lesions attributable to Ascaris or lungworm larval migrations.

Bacteriological examination of consolidated areas of lung was negative.

The numbers of lungworms found in the left lung and the intermediate lobe of the right lung of each pig are given in Table I. All worms were sexually mature, although the females, which measured up to 3.5 cm. in length, had not attained their full size.

Histological Appearance

The histological abnormalities in the lungs were confined to the macroscopically affected parts and may be conveniently described under the following headings: (a) bronchi and bronchioles, (b) emphysema, (c) consolidation.

Bronchi and bronchioles. The location of lungworms in the bronchial system was seen clearly in sections of affected lung, which is shown diagrammatically in Fig. 4. The gravid middle and hind parts of the lungworms were present in the small bronchi (Fig. 7) while sections of the anterior extremities were seen in the bronchioles (Figs. 5, 6).

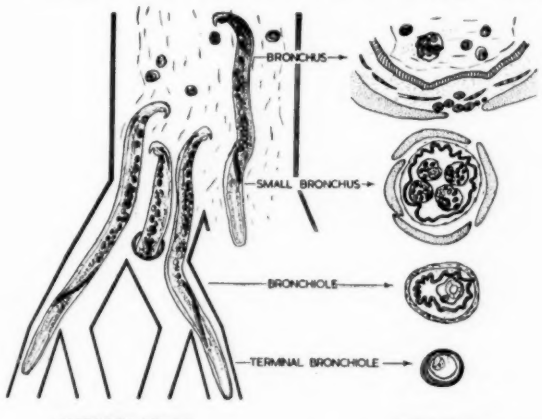

FIG. 4.—Diagram of the location of adult *M. elongatus* in the bronchial system of the pig.

The parasites were surrounded by a mucous exudate containing a few eosinophils, neutrophils, and mononuclear cells and in the larger bronchi this also contained embryonated metastrongyle eggs (Fig. 8). Red and white blood cells filled the intestinal canals of many of the worms.

In bronchi that contained lungworms there was a marked increase in the number of goblet cells in the epithelium and in places practically the whole epithelium was composed of mucus-secreting cells (Figs. 7, 8). This change did not extend to bronchioles containing the anterior parts of lungworms. In places there was hyperplasia of the bronchial epithelium but on the other hand epithelium adjacent to adult lungworms in the lumen had sometimes atrophied. Infiltration of the epithelium with eosinophils, lymphocytes, and plasma cells occurred together with dense cellular infiltration of the lamina propria (Fig. 10). Early lymphoid hyperplasia was seen in the subjacent connective tissue (Fig. 7), and there were numerous mitoses in these foci and in the infiltrating round cells. Hypertrophy of the smooth muscle was present in the bronchioles at this stage of the infection (Fig. 6) and could also be seen in the bronchi, arterioles, and lung parenchyma. Infiltration with histiocytes and non-granular leucocytes together with focal lymphoid hyperplasia occurred in the connective tissue surrounding bronchi, bronchioles, and blood vessels and extended into adjacent alveolar walls to produce increased cellularity and thickening (Figs. 6, 9). Cellular infiltration with eosinophils and round cells sometimes spread to the interlobular septa and subpleural connective tissue.

Emphysema. Pulmonary vesicular emphysema was produced through partial obstruction of bronchioles and bronchi by lungworms and their surrounding mucous exudate (Figs. 5, 6, 11). It did not extend to the interlobular or subpleural tissue. Dilatation of the alveolar spaces in such areas was accompanied by an irregular cellular infiltration of the alveolar walls and perivascular connective tissue with eosinophils and round cells and there was sometimes hypertrophy of the parenchymal smooth muscle. Occasional small "eosinophil" nodules up to 1 mm. diameter were found in affected areas (Fig. 12). These consisted of a central mass of eosinophil leucocytes usually surrounding degenerate parasitic larvae, and an outer zone of endothelioid-type cells. The surrounding lung tissue was partially collapsed and infiltrated with lymphocytes and plasma cells.

Consolidation. Consolidation of the lungs was due to the peribronchial proliferative reactions already noted together with an alveolar response to embryonated lungworm eggs aspirated into the parenchyma (Fig. 13). Eosinophil granulomata as noted in emphysematous areas, were often present and could become confluent. Early interstitial lymphoid hyperplasia and cellular infiltration of the alveolar walls were also pronounced and septal cell proliferation sometimes gave the appearance of epithelialisation of alveoli (Fig. 14). There was an alveolar exudate composed of oedema fluid with varying numbers of eosinophil and polymorphonuclear leucocytes together with lymphocytes, plasma

cells, and alveolar macrophages. The development of multinucleate giant-cells from alveolar macrophages disposed around lungworm eggs and larvae could be traced and numerous free giant cells were found in the alveoli (Fig. 15). Complete phagocytosis of lungworm larvae by formed giant-cells was often seen (Fig. 16).

Enlargement of the bronchial lymph nodes was due to lymphoid hyperplasia and cortical infiltration with round cells and eosinophils. The ileo-caecal lymph nodes showing macroscopic "cysts" contained spaces filled with lymph and dispersed lymphocytes, some of which had degenerated. No significant abnormalities were present in any of the other tissues examined.

Discussion

The localisation of lesions in the caudal extremities of the diaphragmatic lobes and the borders of the lungs has been noted in natural pig lungworm infection (Lewis, 1926; Robertson, 1937; Schwartz, 1952; Dunn *et al.*, 1955; Whittlestone, 1957). Dunn *et al.* (1955) and White (1955) were of the opinion that localisation in the extremities of the diaphragmatic lobes was due to the failure of lungworms to be expelled from these areas and that their persistence represented the residual part of a previous heavy infestation. Examination of the lung lesions produced in the experimental infections described suggested that there was a regular distributional pattern according to the size of the infective dose. This confirmed the predilection involvement of the posterior parts of the diaphragmatic lobes, while the anterior lobes became regularly infected in the heavier infections only. The presence of lungworms in these sites was always associated with distinctive lobular areas of pulmonary emphysema. Consolidation of the lung, however, occurred in the dependant ventral parts of the anterior lobes which is also the usual site for pneumonia of the virus pneumonia type (Betts, 1952).

The manner in which lungworms became selectively distributed in the lungs is conjectural but it seems possible that larvae carried into the lungs in the pulmonary circulation tended to hold to the major and most direct route. In the pig this appears anatomically to be the branches of the pulmonary artery accompanying the main diaphragmatic lobar bronchi which lead to the caudal extremities of the lungs. The disposition of pulmonary consolidation was possibly associated with hypostatic effects in the dependant anterior lobes when they were affected.

Clinical examinations of pigs with experimental lungworm infections revealed that, in the main, coughing was elicited only from those with a heavy infestation, usually sufficient to produce collateral pneumonia of the anterior lobes. Lighter infections were apparently symptomless. It appeared that with lungworms situated in the fine bronchi and bronchioles and where there was no complicating pneumonia, the cough reflex was not readily stimulated.

Conclusions

Using the methods described it was possible to prepare accurate scaled doses of viable infective lungworm larvae and produce experimental infections of pigs where the extent of the infection and of the lesions produced were directly related to the size of the infective dose. There was an apparent distributional pattern of the location of lungworms in the lungs and of the emphysema and pulmonary consolidation produced by them. Clinical manifestations of disease were only produced in heavily infected pigs. The microscopical changes found in the early patent period of the disease differed from those seen in the well-recognised end-stages and indicated a dynamic progressive reaction to the presence of the parasites.

Acknowledgments.—I am grateful to Dr. W. S. Gordon, C.B.E., for his interest in this work, and to Mr. I. H. Pattison for his helpful advice. My thanks are also due to Mr. W. L. Moss for maintaining the earthworm cultures and to Mr. F. H. Summerfield, A.R.P.S., who prepared the photographs.

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AREA ERADICATION PLAN FOR TUBERCULOSIS, 1950
STATEMENT SHOWING THE POSITION AT OCTOBER 1ST, 1958

	Total Cattle on June 4th, 1958 (Provisional Figures) ('000)	Number of Registered Attested Herds	Total Attested Cattle ('000)	Percentage of Total Cattle
GREAT BRITAIN	10,014	202,138	8,231	82
England and Wales	8,196	158,807	6,481	79
England	7,091	120,724	5,435	77
Wales	1,105	38,083	1,046	95
Scotland	1,818	43,331	1,750	96

There were also 3,573 supervised herds needing only one clear test to qualify as attested herds.

Refresher Article No. 12

Newcastle Disease and its Control in Great Britain

BY
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SUMMARY.—The history of Newcastle disease in Great Britain has been reviewed and a description given of the symptoms of the acute and subacute forms. Suggested causes of spread of the disease have been indicated and the legislation in connexion with control has been discussed, together with the practical steps taken in the field to deal with this disease. The diagnostic value of the H.I. Test has been emphasised and the significance of this test explained.

IT was suggested by the Editorial Committee of THE VETERINARY RECORD that fowl pest is of sufficient interest and importance to warrant inclusion in the series of refresher articles published from time to time to assist the practising veterinary surgeon. This article has, accordingly, been written with a view to providing practical information regarding the nature of the disease and its control in the light of experience over a period of years dealing with outbreaks in the field. The term fowl pest actually covers two separate diseases: Newcastle disease and fowl plague; but as the latter has not been recorded in Great Britain since 1922 it is proposed to confine this article to Newcastle disease.

History

Although a disease of fairly recent occurrence in Great Britain, it has, nevertheless, been the cause of serious losses in the poultry industry during the past 10 years. It was first recorded in this country in 1926 when a serious outbreak of disease occurred amongst poultry near Newcastle upon Tyne; this was investigated by Doyle who described it and ascertained that it was caused by a virus. Because of its locality, it was given the name Newcastle disease. Odd outbreaks occurred during the following year, but the disease did not assume serious proportions and ultimately appeared to die out. The lack of spread was no doubt influenced by the rapid course of the disease and the heavy mortality associated with these particular outbreaks. After 1926 the disease was recognised at intervals in various other parts of the world and a further outbreak occurred in England in 1933 when the virus was identified by Dobson. Heavy mortality occurred and surviving stock was slaughtered by the owner, with the result that disease was confined to these particular premises. The Fowl Pest Order was made in 1936 consequent on the extension of the Diseases of Animals Acts to include poultry; the Order gave authority to define and control infected premises and to slaughter affected and in-contact birds.

A disease which had been running in U.S.A. for some years was in 1944 identified as Newcastle disease, and although affected birds showed evidence of respiratory disease with, in some cases, nervous symptoms, the disease generally was very much milder in character than that previously encountered in Europe, and mortality was not nearly so heavy.

The acute form of the disease reappeared in Great Britain in 1947, apparently having been introduced by poultry carcases imported from the continent of Europe; virus was recovered from imported carcases at the Ministry's central laboratory and in the light of this knowledge and the consequent dangers of feeding kitchen waste to poultry, the Disease of Animals (Boiling of Animal Food Stuffs) Order was extended to include waste food fed to poultry. At the same time arrangements were made through the Ministry of Food for imported poultry carcases to be so distributed as to avoid those parts of the country with a dense poultry population. This form of the disease was quite readily recognised; mortality was high, and although spread within the affected flock was rapid, there was little tendency for it to spread to other flocks in the vicinity—affected birds showed obvious symptoms of illness with great depression, and egg production rapidly ceased; profuse greenish diarrhoea was evident with acute respiratory distress and in some cases nervous symptoms, or paralysis. In the latter half of 1947 considerable spread occurred, mainly through markets having become involved or through the medium of poultry dealers and poultry slaughterers.

Scotland had been relatively free from disease and in August, 1947, the movement of live poultry into Scotland and the northern counties of England from the remainder of England and Wales was prohibited by order. This measure prevented the spread to the northern part of Great Britain, and by 1948 outbreaks had diminished very considerably in the rest of the country.

Slaughter of affected flocks was carried out under the Fowl Pest Orders, followed by disinfection of the infected premises, and restrictive notices were served on owners of contiguous flocks. This policy appeared to be effective, but there was a considerable increase in 1949 when the disease appeared in Scotland, mostly in the Islands off the West Coast. A total of 203 outbreaks was distributed as follows:—

Outer Hebrides ...	116	*Mainland West Coast*	17
Inner Hebrides ...	34	*Bass Rock (Firth of Forth)*	1
Orkneys	26	*Mainland East Coast*	6
Shetlands	3		

The considerable number of outbreaks on the Islands suggested the involvement of sea birds, either as mechanical carriers or by themselves becoming infected, and virus was, in fact, subsequently recovered from wild birds submitted to the

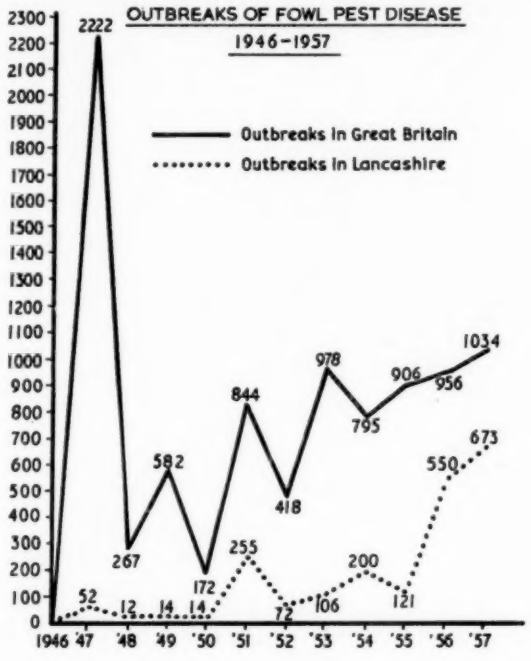
Ministry's laboratories at Weybridge and Lasswade. Cormorants and Gannets were involved.

In 1948 there was evidence that a milder form of disease was present in Great Britain, when birds which had been sent to a laying trial from a hatchery, gave positive reactions to the haemagglutination inhibition test. These birds did not show any clinical symptoms of Newcastle disease, egg production was not affected, and there was no unusual mortality. Other birds at the premises of origin were tested and gave positive reactions, indicating that they had at some time been affected with the disease, though again no clinical symptoms were evident. Enquiries indicated that young chicks at the hatchery had apparently been previously affected with coryza and accordingly it was considered that disease may have existed for some time on the premises implicated.

In October, 1950, after a period of 5 weeks in which no case of Newcastle disease had occurred, an outbreak was confirmed in West Suffolk which gave rise to a large number of cases. This was the first occasion in any series of outbreaks in which the symptoms presented were consistently subacute. The chief symptoms were a sudden drop in egg yield, often accompanied by respiratory distress and coryza. There was a low mortality in adults, although an infected group of young birds had a mortality rate of up to 40 per cent. The H.I. test now came to be widely used to confirm Newcastle disease and the policy of slaughtering affected birds and in-contacts was continued. A check by the use of the H.I. test on birds produced from this and other hatcheries indicated that this form of disease had a very limited distribution. Nevertheless, this mild form has apparently occurred subsequently, but has been dealt with as for the subacute type (*vide infra*).

In October, 1951, Newcastle disease was diagnosed at Longton, south of Preston, when over 60 outbreaks occurred within 4 weeks. These outbreaks were in an area of dense poultry population, and to check the spread of disease restrictions were imposed prohibiting the movement of any poultry out of the area which had been defined by special order. By the end of 1951, 204 outbreaks had been confirmed in this part of Lancashire but the position improved at the turn of the year and disease faded out in the early months of 1952. It was then fully realised that the subacute form of the disease, with its great infectivity, could become a very serious menace in a county such as Lancashire with a recorded poultry population of over 7,000,000 in addition to a considerable number of unrecorded (agricultural returns are only submitted in respect of holdings of one acre or more). Outbreaks occurred from time to time in various parts of the country, Lancashire and East Anglia being the areas most severely affected, and despite vigorous application of the slaughter policy the disease continued to flare up in Lancashire during the winter months reaching its peak in November and December. The incidence of disease from 1947 up to the end of 1957,

(a) in the country generally, and
(b) in the county of Lancashire, is shown in Fig. 1.



Diagnosis

The acute form of the disease is fairly readily recognisable clinically, and has been successfully dealt with by means of the slaughter policy. The subacute form, however, presents considerable difficulties and quite frequently diagnosis by clinical examination alone has not been possible. In such cases the H.I. test is extremely valuable, blood samples from suspected birds being submitted to the Ministry's laboratories either at Lasswade or Weybridge. The clinical picture in this subacute form of the disease can vary considerably, and in some cases, particularly where growing stocks are involved, there may be little in the way of clinical evidence. Mortality is generally low, but it may be fairly high in young birds with, in some cases, nervous symptoms in addition to evidence of respiratory distress. The nervous symptoms can, in extreme cases, resemble those of crazy chick disease and epidemic tremor. In older birds no matter how variable the symptoms, a constant feature is a sudden and spectacular drop in egg production. This sudden drop is usually preceded by the laying of misshaped, soft-shelled or shell-less eggs. There is generally evidence of respiratory trouble in the way of sneezing or coughing, with, in some cases, a slight rattling noise in the throat. There may be some watery discharge from the eyes and nostrils and a greenish diarrhoea is evident. Affected birds are less inclined to feed and generally appear dull and listless.

The main diagnostic features are :—

1. The sudden drop in egg production.
2. Coughing or sneezing with the throat sound referred to above. (This is more apparent at night when the birds are undisturbed.)

3. The greenish diarrhoea.
4. Low mortality.

Whilst the drop in egg production occurs in other respiratory diseases it is generally less spectacular than in the case of Newcastle disease.

It is not possible to distinguish the respiratory diseases of poultry by clinical examination. Infectious laryngotracheitis, coryza, chronic respiratory disease, infectious bronchitis and sometimes fowl pox may all show symptoms that might be associated with subacute Newcastle disease; therefore it is necessary to exclude the possibility of Newcastle disease by the application of the H.I. test on flock basis before proceeding to further differential diagnosis.

Post-mortem Findings

There is considerable variation in the nature of the lesions found on *post-mortem* examination. In some of the very mild cases little or nothing abnormal is found. The lesions fairly commonly seen are an excess of mucus in the trachea with slight congestion of the mucous membrane, some oedema of the lungs, the air sacs may present a cloudy appearance and small haemorrhages may occur in the heart fat, in tissues above the sternum, in the ovary, in the mucous membrane of the proventriculus and occasionally in the musculature. Free egg yoke may be found in the peritoneal cavity.

Spread of the Disease

Newcastle disease virus is highly infectious and passes very readily to contact poultry. There is ample evidence that it is easily carried in crates and vehicles and on the hands and clothes of people who have handled infected birds. Ground vermin no doubt act sometimes as mechanical carriers. There has been some speculation about the part played by wild birds, and while there is little concrete evidence that they are an important factor it seems likely that they can carry the infection particularly when they gain access to feed troughs in infected units from which they move to contiguous flocks.

There is some evidence that the virus may be carried short distances by the wind. When portions of infected carcases are included in kitchen waste this material is highly dangerous.

Control Measures

The policy of the Ministry is one of eradication of the disease by compulsory slaughter of all the birds in infected flocks, together with the imposition of movement restrictions where necessary, and the prohibition of importation of live poultry, hatching eggs and poultry carcases from countries where fowl pest exists. There can be little doubt that this policy has resulted in the elimination of the acute form of the disease. The outbreaks which have occurred subsequent to 1950 have mostly been of the subacute form and these have proved much more difficult to control. Despite the great infectivity of this disease, symptoms, as previously pointed out, are sometimes not readily recognisable: but, although outbreaks in recent years have occurred at disturbingly high levels in the autumn and winter in some parts of the country, the spread of the disease is more effectively controlled by the slaughter policy than can be achieved by any other procedure. Further, the slaughter policy has the support of the poultry industry generally.

The main instrument in the control of the disease is the Fowl Pest Order of 1936 as amended by the Fowl Pest (Amendment) Order of 1947 and the Fowl Pest (Amendment) Order of 1947 (No. 2).

These Orders require any person having in his possession or under his charge any poultry or poultry carcases affected or suspected of being affected with fowl pest to give notice of the fact with all practical speed to a police constable for the area. A veterinary surgeon who examines any poultry or carcases affected or suspected of being affected with fowl pest is also required to give similar notice. The Orders prescribe that a veterinary enquiry shall be carried out on behalf of the Ministry, and powers of entry to the premises are given. If a veterinary inspector making an enquiry suspects that fowl pest exists or has within the past 28 days existed on the premises, he is required to serve a notice (Form A)—which prescribes rules to be observed—on the owner or occupier of the premises. This notice declares the premises which are specified in the notice to be an infected place for the purpose of the Fowl Pest Orders. Power is given to slaughter any poultry affected or suspected of being affected, and also any poultry which appear to have been exposed to infection. Compensation is payable to the owner for poultry slaughtered, other than poultry affected with fowl pest. The Orders require the cleansing and disinfection of the premises and of vehicles, utensils, etc., which have been used on the infected premises. Although powers are given to require the owner to cleanse and disinfect at his own expense, the cost of such cleansing and disinfection is usually borne by the Ministry. Provision is also made for the application of movement restrictions (Form B), regarding poultry on any premises which appear to have been in any way exposed to infection.

The Fowl Pest Infected Areas (Restrictions) Order, 1956, applies to the whole of Great Britain and empowers the Minister to make an Order declaring any area to be an area infected with fowl pest, if he is satisfied that disease is prevalent in such an area. When an area is so declared the provisions of this general order will apply to that particular area. These include prohibition of movement of poultry out of/into and within such an area except under licence and subject to specified conditions; and prohibition of the holding of markets, fairs and sales of poultry (except under licence), and of exhibitions or shows in such an area. It contains other provisions designed to prevent the introduction and spread of fowl pest, such as power to close particular footpaths and premises in an infected area; prescribing clothing to be worn by chick-sexers together with measures for personal disinfection, and requiring cleansing and disinfection of vehicles used in an infected area for the conveyance of poultry, or by egg packing stations for conveyance of eggs.

There is also a provision regarding the disposal of manure and refuse from poultry slaughterhouses and of refuse from egg packing stations.

The Live Poultry (Restrictions) Order, 1957, prohibits the movement of poultry, day-old chicks or hatching eggs into Scotland from other parts of Great Britain. It also prohibits the movement of poultry from any premises to which there has been movement of poultry or poultry carcases (apart from carcases for consumption on the premises), during the preceding 28 days. The Order regulates the holding of poultry exhibitions and shows, and gives authority for the licensing of such shows by local authorities; requiring the person to whom a licence is issued to maintain proper records in respect of birds entered. It prohibits the holding of sales of poultry except under authority of a licence issued by the local authority and prescribes the conditions under which such licences may be issued. Local authorities are authorised to grant licences for sales of poultry for immediate slaughter, and for sales of store poultry (except during the period October 1st to December 31st).

The Poultry and Hatching Eggs (Importation) Order of 1947 and Amendment Order of 1952 prohibits the landing in Great Britain of poultry or poultry eggs intended for hatching if they have been brought from a prohibited country, and prescribes the conditions under which such importation may take place from specified countries which are not prohibited countries.

The Poultry Carcases (Landing) Order, 1955, prohibits the landing in Great Britain of poultry carcases that are not fully cooked from countries outside Great Britain, except as defined in the Order.

The Live Poultry (Movement Records) Order, 1954, requires poultry dealers and auctioneers to keep records of purchases and sales of poultry, and also, in the case of poultry dealers, of day-old chicks.

The Poultry Pens, Fittings, and Receptacles (Disinfection) Order, 1952, requires the cleansing and disinfection of pens, fittings and receptacles used in connexion with markets or shows, and prescribes the manner of such cleansing and disinfection. It also requires the notification by any person of intention to hold an exhibition or a show of poultry.

The Poultry, Premises, and Vehicles (Disinfection) Order, 1956, empowers an inspector of the Ministry of Agriculture, Fisheries and Food, or an inspector of a local authority, if he considers it necessary, for the purposes of preventing the spread of disease, to serve notice,

1. on the occupier of premises used for the slaughter of poultry or for the temporary detention of live or dead poultry or poultry feathers;
2. on the owner or person in charge of a vehicle used for conveyance or exposure for sale of live or dead poultry or poultry feathers;
3. on the owner of person in charge of equipment used for such poultry or feathers,

requiring him to cleanse and disinfect the premises, vehicles or equipment.

When such notice is served, the premises, etc., required to be cleansed and disinfected may not be used in connexion with poultry, poultry carcases or feathers until the work has been carried out to the satisfaction of the officer serving the notice. Further, if the owner fails to comply with the notice, the Ministry or local authority are empowered to cause the necessary work to be done and to recover the cost from the person on whom the notice was served.

Procedure

Suspected cases reported to the Ministry's divisional office are almost invariably dealt with on the same day, unless the report is received late in the day; and the person reporting is warned that no movement of live poultry or carcases, eggs, utensils, etc., must take place pending the arrival of the veterinary officer. Careful inspection of all birds on the premises is carried out and the symptoms noted. *Post-mortem* examination of several affected birds is made to assist in arriving at a diagnosis and if a decision that fowl pest exists on the premises can be arrived at on the evidence available, the findings are reported to a veterinary officer at Headquarters, when slaughter authority may be given. If disease can be confirmed on clinical grounds slaughter is usually carried out the same day. Otherwise, a number of samples of blood are taken, as far as possible from the birds in which the disease was first noted.

If the veterinary officer has grounds for suspecting that fowl pest exists or has existed on the premises within the past 28 days, a notice (Form A), under the Fowl Pest Order of 1936 is served on the owner, and this is so worded as to cover the whole of the holding on which the poultry are kept. This notice must be handed to the owner or his representative and, at the same time, attention is drawn to the rules regarding the infected premises. Confirmed outbreaks are notified to the egg packing station used by the owner, and no eggs are collected from the premises until permission is given by the veterinary officer. This permission is given conditional on the eggs being dipped in a 4 per cent. solution of washing soda, and the outside of the egg boxes wiped with a similar solution. Slaughter of all but the smaller flocks is carried out at night. The birds at this time are easier to handle and the work can be carried out more expeditiously with less disturbance of litter, etc. Slaughter is supervised by a veterinary officer and immediately on completion, poultry houses, litter and carcases are soaked with disinfectant, applied by use of a mechanical pressure sprayer.

Carcases are usually disposed of by burial on the infected premises, but on low lying land where water will be encountered a few feet below ground surface, or on rocky ground, it is necessary to resort to burning. This, whilst efficient, is very much slower and more time consuming in the way of supervision; by utilising excavators of suitable type, burial can be completed in a matter of a few hours. There are premises in some urban areas where neither burial nor cremation is possible. In these, carcases are disposed of in the nearest local authority incinerator, adequate precautions being taken in the way of disinfection before and during

conveyance of the carcases to the place of destruction.

During all investigations rubber waterproof clothing and boots are worn and every precaution taken regarding personal disinfection.

For carrying out the disinfection of premises, etc., mechanical pressure sprayers are very efficient; there are two very good types which give a nozzle pressure of 300 lb. per square inch. Two types of excavators have been extensively used, a back acting mechanical navvy which is excellent for disposing of the larger flocks, and a mechanical digger, mounted on a tractor, which is used for flocks of about 1,000 birds. This latter machine is very mobile and travels on its own wheels at about 15 m.p.h.; it also has a bulldozer at the rear which is of great assistance in filling in the pit. Excavators are disinfected by a high pressure pump before leaving the infected premises.

After disposal of the carcases preliminary disinfection of the whole of the premises is carried out and this consists of soaking all poultry houses, litter, equipment, etc., as far as practicable with an approved disinfectant at the appropriate strength. Litter and manure are closely stacked to ensure heating, and the outside of the dump is sprayed with disinfectant. The poultry houses are then washed down by use of the power pumps, scraped and scrubbed clean, and finally sprayed with disinfectant. Sacks, feeding stuffs and clothing are fumigated by the use of formaldehyde gas. As a general rule, Form A restrictions are removed from infected premises one month after completion of disinfection, but this period may vary, depending on the proximity of recent outbreaks of disease.

Poultry flocks contiguous to, or in close proximity to the infected premises are placed under restrictions, a notice, Form B, under the Fowl Pest Order of 1936 being served on the owner. This prohibits the movement of poultry to or from the premises except under licence.

Flocks immediately contiguous or in close proximity to the infected premises are usually slaughtered with a view to minimising the spread of infection, and samples of blood from all such flocks are submitted for the H.I. test with a view to ascertaining whether disease exists or has existed.

Contiguous and contact flocks which have not been slaughtered are inspected and blood samples taken at intervals of 10 days. All other flocks within a quarter of a mile of the infected premises are similarly inspected and blood samples submitted for the H.I. test.

The H.I. Test

The observation of Burnet (1942) that Newcastle disease virus causes agglutination of suspensions of red blood corpuscles of many species and that this haemagglutination is specifically inhibited by immune sera forms the basis of the H.I. test.

Since the reintroduction of Newcastle disease into Britain in 1947 extensive use has been made of the H.I. test as an aid to diagnosis, whilst the test is a much less sensitive indicator of antibody than virus neutralisation, because of its cheapness and simplicity it has been possible to apply it as a routine in suspected outbreaks. The demonstration that samples of sera from a suspected flock will inhibit agglutination of the red blood corpuscles by Newcastle disease virus is interpreted as evidence of past infection. Some birds may continue to give a positive reaction for many months. Consequently it is not possible to determine from the H.I. test when a reacting bird was infected. The test does not give any indication of the infective state of the bird.

The Ministry's Policy

In Great Britain the policy of slaughter with compensation is considered to be the only method of achieving good control and eventual eradication. While the disease continues to be troublesome in some counties in England a good measure of control has been achieved; *e.g.* in 1957, 33 counties in England and Wales did not experience fowl pest. Since 1947 the approximate annual cost of the stamping out policy has been £600,000; this includes compensation for birds slaughtered, hatching eggs destroyed and disinfection expenses.

The only alternative at present is the widespread immunisation of the national flock. Killed vaccines do not give a durable immunity and the live vaccines which have been used widely in almost all countries that have experienced Newcastle disease give a good immunity for some months but under that system revaccination of breeding fowls is necessary. It will readily be appreciated therefore that any system of control other than a "stamping out" policy means that the industry would have to live with the disease permanently.

If the disease was uncontrolled and 10 per cent. of flocks became affected it has been estimated that losses might reach a figure of £3,600,000 per annum. Even in a system of control by the use of a serviceable vaccine the costs would be high and recurrent —probably well over a £1,000,000 per annum. It should be appreciated that the slaughter policy applied to fowl pest presents greater difficulties than it does when applied to foot-and-mouth disease. Movement restrictions are much less readily enforced. Poultry of all ages including day-old chicks, eggs, poultry carcases and feathers may carry infection, and crates and boxes used in transport may become contaminated. With larger animals vehicles certainly require to be controlled but with poultry the vehicles will include small lorries and motor cars and the containers may change hands as readily as the poultry. In consequence of this difficulty various regulations have been made applicable as the need for them becomes apparent. For example, a scheme for the definition of clean areas into which movement of poultry was prevented was applied for a period—this certainly succeeded in confining outbreaks—subsequently the present infected area restrictions scheme has replaced it. Again, when it became apparent that the activities of poultry slaughterers were involved in the spread of disease (crates, clothing, etc.) the Poultry Premises and Vehicles (Disinfection) Order, 1956 was made.

Acknowledgments.—In the preparation of this article much valuable assistance and advice has been received from my colleagues in the field staff, at headquarters Tolworth, and at the Central Laboratory, Weybridge. This is gratefully acknowledged.

Hypomagnesaemia in Calves in Nigeria

BY

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SUMMARY.—Three cases of hypomagnesaemia in calves in Nigeria are reported. The symptoms and analytical data accord well with those reported for calf hypomagnesaemia in more temperate climates. Grass tetany in adult cattle has not been found in the cases studied.

Introduction

A VAST amount of literature has been published on hypomagnesaemia in temperate climates which has been adequately summarised in reviews by Russell (1944), Allcroft (1954), and Blaxter and McGill (1956). Blaxter and Wood (1952) showed that the availability of dietary magnesium for calves is low and estimations of this availability from the balance trials of Blaxter and Rook (1954) showed that only 30 to 50 per cent. of the dietary magnesium was available for the demands of the animal body. The low availability arises from large faecal losses and the losses due to unabsorbed digestive juices. These authors showed that hypomagnesaemia in calves was a simple dietary deficiency, there being a slow fall in the serum magnesium concentration with a concomitant depletion of the bone magnesium content. It may take 40 to 50 days to deplete the skeletal magnesium reserve and it is a depletion of the magnesium ion concentration in the fluid bathing the neuromuscular junctions and synapses which is responsible for the tetanic syndrome (Blaxter, Rook & MacDonald, 1954). On the other hand hypomagnesaemia in adult cattle in temperate climates is not a simple dietary deficiency but a more complex metabolic disorder (Blaxter & McGill, 1956).

During the year 1956-7 at Vom, 3 cases of hypomagnesaemia have been recorded in bucket-fed calves at the Livestock Investigation Centre. Vom is situated in the Plateau Province in Northern Nigeria at over 4,000 feet altitude; it has a rainy season lasting from April to October each year when the rainfall is approximately 75 inches. The temperature rarely rises above 85° F. in the dry season and rarely falls below 50° F. in the rainy season. The cattle at Vom are relatively healthy and considering the fact that these cases of hypomagnesaemia occurred where the animal husbandry is somewhat better than that encountered with the nomadic Fulani herds, it is thought worth while recording the existence of calf hypomagnesaemia in view of the possible occurrence of this disease in other parts of Nigeria.

Clinical Cases

Case No. 1. Four-month-old female Zebu calf (No. 3280) weighing 156 lb. at 3 months of age.

The first symptoms observed were a stiffness of gait followed by a nervous twitching of the limbs. These symptoms persisted for 2 to 3 days when they became more acute. There was grinding of the teeth, frothing at the mouth, slight protrusion of the eyeballs, and eventually convulsive fits. There appeared to be sensitivity to noise and death occurred within a further 48 hours. The only *post-mortem* signs were ascites, a mild enteritis, and nephritis.

The normal serum magnesium for a calf of this age at Vom is 2.30 ± 0.16 mg. per 100 ml. (Peers, unpublished data). This figure is comparable to these quoted by Jones (1943), of 2.76 ± 0.46, and Garner (1948), of 2.60 ± 0.04 for mean serum magnesium levels determined in the tropics. Table I indicates the progressive nature of the disease.

TABLE I

SERUM MAGNESIUM AND SERUM CALCIUM CHANGES IN CALF NO. 3280

Age (Weeks)	Serum Mg. (mg. per 100 ml.)	Serum Ca (mg. per 100 ml.)
5	2.36	13.4
9	1.58	11.7
14	1.20	11.8
15	0.80	11.3

Treatment was instituted when the serum magnesium level had fallen to 1.20 mg. per 100 ml.; magnesium oxide was given daily by mouth with no effect and injections of magnesium sulphate failed to relieve the condition. Chloral hydrate in small doses helped to relieve the severity of the fits.

Case No. 2. Three-and-a-half-month-old male Zebu calf (No. 3320) weighing 156 lb. at 3 months.

The clinical symptoms were irritability, nervousness, and anorexia followed by muscular tremors. The animal was destroyed at this stage due to its refusal to eat or drink. No lesions were seen on *post-mortem* examination. Apart from milk there was an absence of ingesta in the alimentary canal.

A blood sample taken on the day the calf was destroyed showed a serum magnesium value of 1.38 mg. per 100 ml. and a serum calcium figure of 9.2 mg. per 100 ml.

Case No. 3. Two-and-a-half-month-old Zebu calf (No. 3339) weighing approximately 130 lb.

The clinical signs were anorexia, grinding of the teeth, muscular tremors, and tetany of the extensor muscles. The symptoms were mild for 24 hours and then became severe during the next 24 hours; on the third day the tetanic spasms became so severe that the calf was destroyed. No abnormalities or lesions were seen on *post mortem* except the presence of a few cysts of *Cysticercus bovis*.

Serum magnesium and calcium values were determined on 2 samples taken on the second day and the results gave mean values of 0.91 mg. per 100 ml. for magnesium and 6.3 mg. per 100 ml. for calcium.

Discussion

The recognition of the existence of hypomagnesaemia at Vom occurred in the course of an investigation in which calf No. 3280 was one of a group of animals from which monthly blood samples were taken in order to establish a "normal" blood chemical picture in the Zebu animal. Serum mag-

nesium determinations were carried out on the blood samples as a routine and the progressive drop in the serum magnesium level in this particular calf was noted.

Calves reared at Vom are bucket-fed on whole milk until weaned at 6 months of age. A supplement of poor-quality hay and a minimal amount of concentrates are gradually introduced into the diet from the age of 3 weeks onwards. In the 3 cases reported above, the calves persistently refused to take these supplements, and in all 3 cases the *post-mortem* findings showed only milk in the alimentary canal. Since the identical ration was fed to other calves in a group of 24 animals which did not show any depletion of the serum magnesium content, it is considered that the dietary magnesium supplied was adequate. However, Duckworth (1939) pointed out that too much emphasis should not be placed on the dietary adequacy since calves can become hypomagnesaemic on a milk diet alone, whereas partial replacement of the milk by cereals containing an equivalent amount of magnesium can prevent the condition.

With calf No. 3280 the first symptoms appeared at 14 weeks of age when the serum magnesium level was found to be 1.20 mg. per 100 ml. This is in agreement with the observations of Blaxter (1954), who found that no clinical symptoms occurred in the United Kingdom until serum magnesium values below 1.40 mg. per 100 ml. were reached. The drop in serum magnesium is usually gradual (Blaxter & McGill, 1956), and it was therefore somewhat surprising that 5 days following the value of 1.20 mg. per 100 ml. the serum magnesium value had dropped to 0.80 mg. per 100 ml. However, cases have been reported by Stewart (1954), and by Allcroft (1954), where the serum magnesium level has fallen precipitously under certain conditions.

According to Blaxter (1955), it is unlikely that a serum magnesium value of 1.38 mg. per 100 ml. would cause clinical hypomagnesaemia, yet clinical symptoms were observed with the second calf (No. 3320), which accorded with the early stages of magnesium deficiency. It is recognised that in the final stages of convulsive attacks the serum magnesium is often increased due to the release of magnesium into the blood stream by rapidly contracting muscles, and also that blood samples taken immediately prior to death or at *post-mortem* examination can give high values (B.V.A. publication, 1957).

There is a tendency for low serum magnesium to be associated with low serum calcium in grass tetany —see Nicholson and Shearer (1938) and Nolan and Hull (1941). However, the work of Dryerre (1932), Allcroft and Green (1934), and Hopkirk *et al.* (1933) has shown that low calcium values are not always found in hypomagnesaemia. It is of interest to note that in calf No. 3280 the serum calcium did not drop appreciably whereas in the other 2 calves the serum calcium values were significantly lower than normal.

On the whole these results and figures agree with those published by other workers in this field and in the case of No. 3320, comparing both the magnesium and calcium figures with those of No. 3339, it is very probable that if the animal had been allowed to live a further day or so, the values would have dropped still further and, in fact, the animal was destroyed somewhat prematurely.

In Fulani herds in Nigeria all calves are suckled and supplementary feeding is not a normal practice. The Fulani, however, feed "natural salt" preparation, derived from lake or river-bed deposits, in which the magnesium content is of the order of 0.5 per cent. Animals given free access to such preparations will only consume approximately 15 g. per day which is equivalent to about 75 mg. magnesium per animal per day (Peers, unpublished data).

In adult animals in the United Kingdom, hypomagnesaemia has a seasonal incidence with a maximum peak in the spring when the animals are turned out to pasture and a less pronounced peak in the winter (Allcroft, 1947). Early experiments had emphasised this high spring incidence and it was not until Allcroft and Green (1938) recorded its existence in the winter months that the disease was regarded as other than a metabolic disturbance associated with the first flush of spring grass. So far as we can ascertain there are no reports of grass tetany in Nigeria and we can find no seasonal variations in the serum magnesium values of adult cattle at the Vom stock farm (Peers, unpublished data).

In the light of these reported cases of hypomagnesaemia in bucket-fed calves, it may well be that with the suckling calf fed no supplementary rations, the existence of hypomagnesaemia is more widespread than previously thought although it seems unlikely that the disease is of economic importance in adult cattle.

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Foot-rot in Sheep

A Comparison of Treatment with Formalin, Oxytetracycline* and Chloramphenicol†

BY

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SUMMARY.—1. A comparison of treatments for contagious foot-rot in sheep between 10 per cent. formalin as a bath, oxytetracycline ointment and chloramphenicol 10 per cent. tincture, showed a slight advantage for chloramphenicol 10 per cent. tincture in the early stages of treatment, and a trend in favour of terramycin in the middle stages. At the end of the period of observation there was little to choose between the 3 methods of treatment.

2. The costs of treatment with the 3 substances are compared, and some physical factors associated with them are discussed. Statistical analysis of the results, however, showed no significance between any of the treatments, and the recorded results could be explained by chance fluctuations.

3. The concentration of formalin in the foot-bath remained high during the winter months when these observations were made.

IN recent years claims have been advanced by several investigators that as a means towards the final eradication of the common contagious form of foot-rot, as described by Beveridge (1941), one or other of the three therapeutic agents, formalin, oxytetracycline and chloramphenicol, has superior properties (Sambrook, 1955; Stewart, 1954; Harriss, 1955; Littlejohn, 1955). Advocates can also be found for other substances including copper sulphate, the sulphonamides, antimony trichloride and also for preparations of undisclosed formulae. A discussion of the problem at the Annual Congress of the British Veterinary Association (1956) adjourned with an air of indecision, but the feeling was voiced that a controlled comparison of treatments between formalin, oxytetracycline and chloramphenicol would do much to help clear up the question.

This paper describes a trial carried out under field conditions to determine if any of the three substances—formalin, oxytetracycline or chloramphenicol possessed properties which made it superior for use in the general treatment of foot-rot.

The Flock

The flock available for the trial consisted of 244 pure-bred Clun ewes, 117 pure-bred Wiltshire Horn ewes, 40 pure-bred Hampshire Down ewes and 31 assorted cross-bred ewes. Prior to tupping in October 1956, none of these ewes showed visible evidence of foot-rot, after a dry, warm summer. The early part of the winter was wet and cases of foot-rot became so numerous that towards the middle of January, when the ewes were already 3 months pregnant, it was necessary to carry out mass treatment.

The muddy conditions of the sheep yards and approaches prevented their use, whilst climatic conditions would have entailed handling wet sheep continually. The problem was solved by carrying out the examination and treatment in an open shed fitted with a raised battened floor. This provided facilities for partially drying the sheep and for having reasonably clean feet with which to work. Space, however, was limited and it was necessary to examine one third of the flock without regard to breed, on each of 3 successive days. The middle date of January 23rd, 1957 was adopted as the average for commencement of treatment.

Methods

All ewes were turned up and their feet examined and trimmed. Any suffering from obvious foot-rot or which showed evidence of the disease at the time of trimming were drafted out for further attention. The remainder were passed through a 10 per cent. formalin foot-bath, spending approximately 1 to 5 minutes in it, and walked out on to pasture rested from sheep for at least a month.

The number of sheep found affected was 76 ewes. The treatment of the 3 groups was as follows:—

Group 1—23 sheep. Affected feet were pared with foot-rot shears and with a knife where necessary, after which each sheep was held for 5 to 10 minutes in a bath of 10 per cent. formalin freshly prepared for this group.

Group 2—31 sheep. After paring, each affected foot was liberally smeared with Terramycin Topical Ointment. Unaffected feet were similarly treated with a less liberal amount of oxytetracycline ointment. The sheep then passed directly on to clean pasture.

Group 3—22 sheep. After paring, affected and unaffected feet were painted with 10 per cent. chloromycetin tincture. Treated sheep then passed directly on to clean pasture.

When each affected foot was being pared, it was assessed at 3, 2 or 1 points according to the severity of disease process encountered. This assessment was made because it could happen that all affected feet in any one group might be only slightly affected, whereas another group may be burdened with severe infections only. The assessment had to be made on visual evidence alone, but in general, 3 points were recorded against severe lesions involving considerable separation and overgrowth of the hoof, under-running of the sole, exposure of moist bleeding laminae and a high degree of heat and tenderness.

* Terramycin Topical Ointment: Pfizer & Co., Folkestone.

† Chloromycetin 10 per cent. Tincture: Parke, Davis & Co., Ltd., Hounslow.

Two points were awarded to feet with lesions of moderate severity, including "cheesy" soles with moderate under-running, slight separating of the wall of the hoof including areas of sinus formation, and moderate heat and tenderness.

One point was counted where lameness was slight or absent, but where there was evidence of activity, where small pockets of infection were uncovered or where the interdigital cleft alone showed evidence of infection.

Further Treatment

At intervals of approximately 10 days all 76 affected ewes were brought in together. Their feet were again pared and examined and an assessment made of progress towards recovery. Each treated foot was classified as:—

(a) H = healed,
(b) B = better or healing, and
(c) A = active.

Where lesions persisted, 1, 2 or 3 points were allotted as above. This information was passed to an assistant who recorded the progress and then informed the operator of the treatment category of the affected ewe. This procedure, coupled with the fact that it was impossible under the then prevailing weather conditions, to detect any evidence suggesting the nature of the previous treatment, meant that the judgment of the adjudicator was not biased in favour of any one method.

At the second and subsequent treatments the necessity to economise in oxytetracycline and chloramphenicol, precluded the use of these antibiotics for unaffected feet. The unaffected fore-feet were then immersed in a jar of freshly prepared 10 per cent. formalin, and unaffected hind feet were swabbed with the same material. This was more economical of labour than attempting to stand the ewe up and immerse her hind feet in jam jars. These sheep then passed directly on to clean pasture without passing through a foot-bath.

In Table I the results of all the assessments are summarised and in Table II the "points" awarded for severity of each examination have been expressed as a percentage of the original marking.

The results of this trial suggest that of the 3 substances selected for test, the quickest results may be obtained with a 10 per cent. tincture of chloramphenicol, when account is taken of the severity of the treated lesions. The trend in favour of chloramphenicol by the end of the period following initial treatment was, however, not maintained and by the end of the period following the second treatment, oxytetracycline ointment showed a significant superiority both in numbers of feet cured and in reduction of severity.

When the sheep were examined 10 days after the third treatment, the differences were slightly in favour of the antibiotics though the margin between them and formalin was scarcely significant. By the end of still another 10 days, or 43 days from commencement of treatment, no significance was determinable between any of the 3 methods of treatment.

TABLE I
PROGRESS OF HEALING IN TREATMENT GROUPS

Treatment ...	Formalin 10 per cent.						Terramycin ointment.						Chloromycetin 10 per cent. tincture					
Sheep in group	23						31						22					
Number of feet involved at 23.1.57 ...	34						41						27					
Classification	A	%	B	%	H	%	A	%	B	%	H	%	A	%	B	%	H	%
4.2.57 ...	13	38	7	21	14	41	12	29	17	41	12	29	8	30	11	41	8	30
14.2.57 ...	4	12	12	35	18	53	4*	10	7	17	31	74	5*	18	7	25	16	57
25.2.57 ...	4	12	3	9	27	79	2*	5	8	19	33	77	2*	7	4	14	23	80
6.3.57 ...	1	3	1	3	32	94	2	5	2	5	39	91	1	3	2	7	26	90

A = Active lesions. B = Healing lesions (better). H = Healed.
Note.—* Previously unaffected foot found to be infected.

TABLE II
COMPARISON OF THE PERCENTAGE OF FEET REMAINING INFECTED AFTER SUCCESSIVE TREATMENTS

Treatment ...	Formalin 10 per cent.				Terramycin ointment				Chloromycetin 10 per cent. tincture			
Severity at	A	B	Number of points	Percentage	A	B	Number of points	Percentage	A	B	Number of points	Percentage
23.1.57 ...	—	—	58	**100**	—	—	81	**100**	—	—	66	**100**
4.2.57 ...	26	7	33	**57**	24	17	41	**51**	16	11	27	**41**
14.2.57 ...	8	12	20	**34**	8	7	15	**19**	10	7	17	**26**
25.2.57 ...	8	3	11	**19**	4	8	12	**15**	4	4	8	**12**
6.3.57 ...	2	1	3	**5**	4	2	6	**7**	2	2	4	**6**

A = Lesions still active. B = Lesions healing.

Statistical Analysis

Query: Do the data (as represented by "Table I" and "Table II") suggest that any one of the three treatments is superior (or inferior) to the others for at least part of the time the investigation lasts?

Statistical Treatment: (I) "Table I": 3 x 3 contingency tables were constructed for each date of classification; χ^2's were computed and their probability levels ascertained.

(II) "Table II" 3 x 2 contingency tables, otherwise similar treatment as (I).

Results:

Classification	Table I χ^2_4	approx. Prob.	Table II χ^2_2	approx. Prob.
4.2.57	4·3	36 per cent.	3·3	19 per cent.
14.2.57	4·9	30 ,,	4·6	10 ,,

Conclusions: There is no significant difference between treatments (the data on the other dates of classification are even closer to one another and further statistical analysis is not necessary).

Costings

Ten per cent. Formalin as a Foot-bath

One gallon of formalin (40 per cent.) costs approximately 7/-, and is adequate for about 100 sheep. The use of formalin, therefore, costs less than 1d. per sheep. To keep cost to a minimum it is recommended that the depth of fluid in the bath be kept at the lowest level consistent with completely immersing the foot, otherwise much fluid is lost by splashing and wave motion.

Oxytetracycline Ointment

The cost at 2½d. per foot treated is still the same as that indicated by Harriss (1955). To this must be added the cost of foot-bath treatment for unaffected feet. This would bring the cost of treatment up to 1d. per sheep for sound sheep and 3½d. for sheep with foot-rot in one foot only where the affected foot has been treated with oxytetracycline.

Chloramphenicol Tincture (10 per cent.)

The cost of treating affected feet has been calculated to be 1/- per foot, based on the manufacturer's statement that 2 fluid ounces is sufficient to treat 25 to 30 affected feet. The use of the foot-bath as indicated for oxytetracycline is still necessary and the costs indicated should apply for chloramphenicol on the basis of 1/- per affected foot and 1d. per sheep, making a total of 1/1d. per sheep with only one foot affected.

Observations on the Concentration of Formaldehyde in a Foot-bath During Use, January, 1957

Two gallons of commercial formalin solution (37 to 41 per cent. formaldehyde) were mixed with 18 gallons of tap water in a clean wooden foot-bath 7 ft. 4 in. long and 1 ft. 10 in. wide (measured at base) and fitted with sloping sides. When their feet had been cleaned and pared sheep were passed through this foot-bath at the rate of 20 to 30 per hour. For comparison a bath of similar size was likewise prepared and left open to the atmosphere. At the commencement of the trial and at hourly intervals afterwards, the concentration of formaldehyde was determined in each bath by titration with alkali after oxidation with hydrogen peroxide. On the day of the test, conditions were dry under foot and little extraneous matter was conveyed into the bath by the sheep's feet.

The original concentration of the formaldehyde solutions was 3.5 per cent. w/v. In the bath used for treatment it fell at the end of 3 hours to 3.1 per cent. w/v when 92 sheep had been passed through. The concentration in the control bath remained steady during this period; in fact there was no fall after 24 hours. In a second trial when 76 sheep were passed through the bath in 3 hours, the concentration of formalin fell from 3.9 to 3.8 per cent. w/v, and in a third test after 126 sheep had used the bath the residual solution was 3.7 per cent. w/v.

From the above it is evident that:—

(a) little or no variation due to vapour losses to the atmosphere may be expected for 24 hours from a formalin foot-bath at January temperatures, and;

(b) no great variation, in the absence of gross contamination, was encountered in the strength of a 10 per cent. solution of formalin when used for about 100 sheep. The upper limit has not been determined as under the conditions experienced by us, the limiting factor in treatment is the volume of fluid remaining in the bath after approximately 100 sheep have passed through.

Physical Considerations

Under the very wet weather conditions prevailing at the time of this trial, no undue hardening was observed in feet which had been treated with formalin. This hardening might be a factor of importance under dry conditions.

Whilst adult sheep can be passed with ease through a formalin foot-bath, young lambs have been reduced to a state of near panic and very careful management is required to avoid casualties. The lambs resent the smell of the formalin vapour, become alarmed and splash each other with the solution. This inflames their eyes, and it is only with difficulty and constant vigilance that the animals are passed safely through the bath. It has also been noted that persons working in proximity to formalin baths for long periods may suffer quite severe irritation of the eyes and nose even in open air conditions.

No disadvantageous physical properties are associated with oxytetracycline ointment or chloramphenicol 10 per cent. tincture, though the dye in the latter invariably stains hands and clothing.

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Current Literature

BOOK REVIEWS

Neomycin: Its Nature and Practical Application. By SELMAN A. WAKSMAN. Pp. 412. Baillière, Tindall & Cox, London, 1958. 40s.

Neomycin is a text-book for the antibiotic of that name, which was first isolated in 1949 by S. A. Waksman and his co-workers of Rutgers University. The book is divided into 4 sections covering chemistry, pharmacology, clinical uses, and miscellaneous items respectively. The 30 chapters are written by some 35 authors, who are specialists in their respective subjects.

We are told that neomycin is similar in many respects to streptomycin which is, incidentally, another discovery of the great Professor Waksman. Among the more useful properties of neomycin are its slow absorption from the alimentary tract, and its efficiency against skin infections.

The indications for neomycin in veterinary practice are the subject of 2 chapters. It is recommended in particular for the treatment of bovine mastitis due to Pseudomonas spp., gastro-enteritis, skin conditions, and diseases of the eyes. The possible uses of neomycin as a food supplement are also discussed, although this aspect has not been fully investigated. Nevertheless it is apparent that neomycin has a detrimental effect upon pigs when fed at a dosage of 10 mg. per lb.

This book is a comprehensive survey of all aspects of neomycin, with a bibliography of 691 main references, and it is thereby an essential reference book for all research workers concerned with the field of antibiotics. However, since the veterinary aspects of neomycin did not receive study until 1952 this book has little practical advice to offer to the veterinary surgeon in the field.

Disinfection and Sterilisation. By G. SYKES, M.SC. (LOND.), F.R.I.C. 1st edition, 1958. Pp. 396. Plates 7. E. & F. N. Spon Ltd., London. 75s.

There can be few people who, at some time or another, do not find themselves faced with a problem involving disinfection and sterilisation. In these circumstances it is surprising how few books there have been which deal with these subjects. The possibility exists that this gap in the literature is not unconnected with the many facets of the subjects of which any would-be author would require to have an expert knowledge before he could cover such a wide field.

In recent months there have been 2 books concerned with disinfection and sterilisation, that under review being the more comprehensive. With the publication of this book, it would appear that the gap has been filled. The author brings to his task not only the wide experience he has had in some 30 years of dealing with his subject in the course of his services with a well-known pharmaceutical house, but from the many problems which have been referred to the some 100 or more committees to whose deliberations he has contributed.

Whilst the author would be the last to claim that his book was written specifically for the veterinary profession, there can be little doubt that within the pages of his book he has provided a lot of information having considerable interest to the profession and that he provides authoritative answers to many problems.

There are 17 chapters in the book which is divided into 6 parts. The first part (Chapters 1 to 4) is concerned with the theoretical approach to disinfection and the various methods which are available for testing the many types of disinfectant. Of necessity, a certain amount of the material included within this and other parts of the book are controversial but, whilst the author is careful to present the different viewpoints, he is never afraid to indicate his own preference. The second part of the book (Chapters 5 to 9) covers a description of the varying methods which can be employed in the sterilisation of both liquids and solids.

During the last few years the necessity of having available the larger and larger volumes of sterile air, which are necessary for the production of antibiotics and other products of fermentation, has caused the author to decide this topic justified a part to itself (Chapter 10). The fourth part of the book (Chapter 11) is devoted to problems involved in the killing of viruses and this is followed by a section (Chapters 12 to 16) in which the many types of chemical disinfectants are discussed and their field of application outlined. The final pages (Chapter 17) are reserved for a consideration of all the varying aspects involved in preservation.

Taking the book as a whole, it is difficult to imagine that there is a single topic to which at some time or another Sykes has not turned his attention. It is inevitable that, when faced with the necessity of compressing such a mass of information within the covers of a volume which will be convenient to handle, the author has not been able to say all he might be asked for on every topic. This problem has been solved by the scope of the more than 1,000 references which are given. In the same way, the inclusion of very comprehensive author and subject indices adds to the reference value of this book which appears likely to be the standard reference work on these subjects for many years to come.

Processed Plant Protein Foodstuffs. Edited by A. M. ALTSCHUL. Pp. 955. Many Figures and Tables. Academic Books Ltd., London, 1958. Price 186s.

Science generally is enjoying (?) its own atomic bomb, for the explosive growth of research of the post-war years has left the individual worker feeling ever more remote from the common basic knowledge from which we all begin, and less and less capable of communicating with fellow workers in the different

spheres of enquiry. Many veterinary surgeons may be wondering why a book with the title of that above should be reviewed in their professional journal.

One answer to such a question is that Britain imports nowadays some 800,000 tons of oilseeds or of oilcakes and meals every year. Shall we say that it would provide energy sufficient for 300,000,000 gallons of milk or, used as a protein source, enough for five times that fantastic amount? The reader may amuse himself by turning these figures into their porcine or avian equivalents, but at least he will know why plant protein feeding-stuffs, the subject of the book above, are important to so many people in Britain, either directly or indirectly.

It is as difficult to review this volume, with its virtual 1,000 pages, its 38 authors, and its 3,000 literature references (all but 16!), as it would be to discuss a library: the task can be tackled only in the most general terms.

Let it be said from the start that the worst thing about the book is its price, which will deter many interested people from buying it and using it as a reference book, the only way in which it can profitably be used. Although most of the contributors are American, it appears to be reasonably catholic in its outlook. There is some disproportion in the numbers of references devoted to the different chapters: they range from about 50 in the more modest chapters to the 361 pertaining to the groundnut or peanut, but such differences are to be expected in so ambitious a venture, and no chapter leaves its reader without a reasonable guide to the relevant literature.

After a very brief introductory chapter, the remaining 32 chapters are divided into 2 main blocks, for Part I deals with the general properties of plant proteins whilst Part II describes the attributes of the individual foodstuffs: in chapters the distribution is as 12 to 20; in pages it is roughly 350 to 550. Since the first part contains chapters devoted to (a) proteins, (b) plant proteins, and (c) oil-seed processing and the effects of heat and other technical factors on vegetable proteins, it is clear that the basic science of the subject has not been neglected. Next follow chapters in which the measurement of protein quality, the use of processed plant proteins in animal feeds and the potential uses of such substances are well discussed. A chapter is given to a consideration of chemical sources of nitrogen (*e.g.* urea) as supplements to plant proteins, and another one to the problem of adding synthetic amino-acids to such foods. These are instances where science, with a capital S, has already stepped into the feeding-stuffs business.

Part I thus provides a great deal of information of interest to members of the veterinary profession. Will they profit by it? One hopes so, for in Chapter 6 is given what must be one of the worst examples ever of the results of *modern* scientists and technologists being ignorant of the literature of their own subject. In 1916 the toxicity of trichlorethylene-extracted cakes for cattle was reported in Scotland. In 1927 a similar problem arose in Germany, whilst in 1950, "the information was not well known when renewed interest in trichlorethylene extraction of soya-beans developed in Italy, the United States, and Japan": in consequence there was further trouble with cattle. Such things may easily happen again, for to publish a discovery in a journal nowadays is quite often not to proclaim it to the world but to bury it in an unread volume. The tendency to "read the literature" very lightly is apparent even in this country, though not to the same parochial extent as frequently happened in the U.S.A., where, at least, the problem is now receiving attention. Up to the year 1955 or so, this book seems to have made a wide survey of the literature relating to plant proteins and is thus a good source, not only of factual information, but of references to provide starting points for searches through the published work on many topics.

Part II of the book takes the different plant proteins in turn; soya-bean, groundnut, cottonseed, sesame, sunflower, rapeseed, mustardseed and poppy-seed, linseed, coconut, and palm kernel of the usual oilseed group are followed by chapters on peas and beans, fermentation products, milling products, lucerne meal, microbial protein, algal proteins, inedible oilseed meals (*e.g.* castor), and plant residues —"the lot," to use the current jargon. In each case the general treatment is the same; it begins with an account of the nature of the raw material, follows with a description of the processes used for it, and ends with an outline of the uses of the product. Quite clearly some of the contributors are much more familiar with one aspect than with the others. Thus one cocks an eyebrow at the statement that "a ruminating animal is supplied with a large storage tank called a rumen." Should the general editor have let it pass? Perhaps not, but the reviewer, for one, is not inclined to cavil at the occasional doubtful word or phrase and to ignore the mine of information that is provided. In the Preface the book is said to be addressed "to nutritionists, medical scientists, and students of proteins; to government and health authorities who seek to solve the problem of feeding a growing world population; and to commercial producers and consumers of plant protein foodstuffs." When one reads of starvation in India at the present time and considers the need for the dissemination of such technical information, one's only regret is that the price of this well-published volume may be the major obstacle to its use. As for the hyper-critical, they can always start to write their own books: if they attempt to cover a field of this breadth it will keep them quiet for some years.

Meat Hygiene. By A. R. MILLER, D.V.M., L.I.B. Second edition. Pp. 557. Henry Kimpton, London, 1958. 63s.

The author of this important book is Director of the Meat Inspection Division of the United States Department of Agriculture, and an international authority on this subject. In this edition 3 new chapters have been added, covering Food Poisoning, Chemical Additives, and Ionising Radiation, and the text has been revised with particular regard to the increasing importance of poultry inspection.

The book opens with a brief review of the history of meat hygiene from the Hebraic Laws of Terephah to the Poultry Inspection Act made law in the United States in 1957. Readers of "The Jungle" will remember Upton Sinclair's description of conditions in Chicago packing plants, which is echoed in the report of a special Committee of investigation appointed by the President in 1906. The Committee's findings led to the passing of the Meat Inspection Act in June, 1906, and to the establishment of the Federal Meat Inspection Service.

An early chapter deals with *ante-mortem* inspection and describes some of the diseases which may affect the judgment of the animal before slaughter. After inspection, animals are placed in one of 3 categories, passed, suspect, or condemned, and figures are given for the number of animals condemned in 1956. Unfortunately, the reader is not given any indication of the numbers of animal slaughtered as suspect or the results of *post-mortem* examinations of such animals, and he is left in doubt as to the true value of *ante-mortem* inspection.

The long chapter on *post-mortem* inspection includes a valuable description of the process of slaughtering, and links the various stages with the practical techniques of meat inspection. Identification of carcases with offals in large slaughtering establishments is often difficult, but Dr. Miller indicates how this may be done to the satisfaction of the inspector but with the minimum of disorganisation to the production line. A brief but authoritative description of the carbon dioxide method of stunning is included and will be read with interest by British readers in view of its topical significance.

The lay-out of the section on Pathology is haphazard, and pathological conditions are not grouped in any logical order. This is the least satisfactory part of the book, but the subject is adequately covered in other standard English language works.

Trichinosis merits a chapter to itself and the author attempts to justify his country's policy towards it. In the U.S.A. the public is recommended to cook all pork products thoroughly on the assumption that the parasite may be present, while on the Continent of Europe a vast effort is directed towards the microscopic detection of parasitic lesions after slaughter, with indifferent results. In our own country, the parasite is largely ignored, and occasional outbreaks of trichinosis in the human population, although sensational while they last, are forgotten as soon as possible.

Miller fully recognises the importance of slaughterhouse hygiene, and writes in a practical and comprehensive manner on Sanitation in Plant Operation. Water supply, sewage disposal, rodent and insect control, and slaughterhouse cleansing are covered in the early part of this chapter which goes on to describe the lay-outs of slaughterhalls and workrooms. Various systems of refrigeration, the lay-out of chill-rooms, and the disposal of edible and inedible by-products are thoroughly dealt with.

Meat grading, the adulteration of foods, chemical, and other additives, the preparation of meat and meat food products, chilling, freezing, canning, and sausage preparation also receive detailed treatment. A whole chapter is devoted to ionising radiation, and adequately covers both defence and commercial considerations.

The book is well produced, on good quality paper, and is adequately illustrated. It will appeal primarily to veterinary surgeons and others engaged in food inspection and to post-graduate students who will find it full of valuable and practical information. To other veterinary surgeons and undergraduate students it will serve as a useful reference book and they may well be surprised to learn the scope and interest of the work of their colleagues who specialise in this important aspect of public health work.

Symposium of the Society for Experimental Biology, Number XII. The Biological Rèplication of Macromolecules. Cambridge University Press, 1958. 50s.

Possibly the title of this Symposium is a little forbidding to the layman, but the subject matter, if at times specialised and couched in specialist jargon, contains much that is important and often enthralling.

Basically the Symposium deals with current theories (and facts) on the natural synthesis of large biological molecules such as proteins and polysaccharides. This subject has long been neglected because of lack of precise facts, but the remarkable strides made in the development of analytical techniques such as chromatography, electrophoresis and electron microscopy have resulted in so much exact information on large molecules, *e.g.* the exact amino acid sequences of insulin and of "sickle cell" haemoglobin are now known, that it is possible to divert attention to the processes of biosynthesis.

Probably one of the most important advances in our knowledge of large naturally occurring molecules is the gradual revelation of the properties and functions of the nucleic acids, RNA and DNA. That these play essential rôles in all vital processes of all living cells is now well known and there are dozens of examples in this Symposium. While many of the articles are somewhat technical, one or two are most readable and refreshingly stimulating, particularly that of F. H. C. Crick—"On Protein Synthesis." This is written with a commendable clarity and forthrightness, and it is encouraging to read such definite statements as "*The main function of proteins is to act as enzymes,*" and to realise that there are still those who are not afraid of indulging in reasonable speculation even if the facts are meagre.

The use made of Gamow's "coding" system is particularly interesting in this respect.

Among the other articles of general interest are those on Colicine and Colicinogenic factors, replication of animal viruses and bacterial transformations. All biochemists should read this book and much of the information could and should be included with advantage in student lectures.

News and Comment

THE COLONIAL VETERINARY SERVICE

Promotions: J. W. Macaulay, F.R.C.V.S., Chief Veterinary Research Officer, Kenya, becomes Assistant Director of Veterinary Services, Kenya; M. L. Burdin, Senior Veterinary Research Officer, Kenya, becomes Chief Veterinary Research Officer, Kenya; R. A. Hutchinson, Senior Veterinary Officer, Ghana, becomes Chief Veterinary Officer, Department of Animal Health, Ghana; R. H. Johnson, B.V.SC., M.R.C.V.S., Dip. Bact., Veterinary Education Officer, Federation of Nigeria, becomes Principal Veterinary Research Officer, Federation of Nigeria; I. B. Pullon, Veterinary Officer, Tanganyika, becomes Assistant Director of Veterinary Services, Tanganyika; D. P. P. Scott, M.C., Veterinary Officer, Tanganyika, becomes Senior Veterinary Officer, Tanganyika; J. D. Turton, Senior Veterinary Officer, Ghana, becomes Principal Veterinary Officer, Animal Health Department, Ghana.

Contract appointment: E. C. S. Dawe, ex-D.V.S., Tanganyika, Veterinary Officer, Education, Uganda. (Principal of the Veterinary Training School, Entebbe, Uganda).

New appointments: Miss E. C. Craigie, M.R.C.V.S., B.V.M.S., Scientific Officer, East African Trypanosomiasis Research Organisation, Tororo, Uganda; J. A. Hart, B.SC. (Zoology), Parasitologist, Federation of Nigeria.

XVIth INTERNATIONAL VETERINARY CONGRESS

The preliminary programme and forms of application for membership may be obtained from the United Kingdom member of the Permanent Committee for International Veterinary Congresses, Dr. W. R. Wooldridge, at The Animal Health Trust, 14 Ashley Place, London, S.W.1.

All questions regarding the Congress and all registrations (to be made on special forms) will be dealt with by Dr. Wooldridge. The fees for registration will be : 600 pesetas (£5) for ordinary members of the Congress; 300 pesetas (£2 10s.) for associate members. The publications of the Congress will be sent to persons not attending, but subscribing, for the sum of 400 pesetas (£3 6s. 8.d).

We shall publish some details of the preliminary programme next week.

PERSONAL

Mr. J. A. DeGaris has been transferred from Reading to Tolworth on promotion to Divisional Veterinary Officer.

Mr. J. J. Murdoch has been appointed Veterinary Officer and has taken up station at Chelmsford.

Miss P. E. Logsdon has been appointed Veterinary Officer and has taken up station at Dumfries.

Marriages

ASHWORTH—PICKARD.—On October 18th, 1958, at St. Mary's Parish Church, Kingston, Keith T. Ashworth, B.V.SC., M.R.C.V.S., to Barbara Pickard, B.V.SC., M.R.C.V.S. Present address: Short Hill, Livesey Road, Ludlow.

MACPHERSON—WALLACE.—On October 31st, 1958, at St. Mungo's Parish Church, Alloa. Alasdair M. MacPherson, B.V.M.S., M.R.C.V.S., of Torvaig, Isle of Skye, to Margaret Wallace, M.C.P.S. of Alloa, Clackmannanshire.

COMING EVENTS

November

12th (Wed.). Meeting of the Southern Counties Veterinary Society at the Red Lion Hotel, Salisbury, 7.30 p.m.
Annual General Meeting of the West of Scotland Division at the Veterinary Hospital, Bearsden Road, Bearsden, Glasgow, 2.30 p.m.

14th (Fri.). General Meeting of the Midland Counties Veterinary Association at the Birmingham Medical Institute, 36, Harborne Road, Birmingham, 15, 2.15 p.m.

18th (Tues.). Meeting of the Society for the Study of Comparative Medicine in the Lecture Theatre of the School of Veterinary Medicine, Cambridge, 8.30 p.m.

19th (Wed.). Meeting of the Warwickshire Veterinary Club in the Regent Hotel, Leamington Spa, 8 p.m.

20th (Thurs.). Joint Meeting of the Sussex Veterinary Society and the Mid-Sussex Division of the B.M.A. at the Dudley Hotel, Brighton, 8 p.m.
Annual Dance of the V.V.B.F. County of Ayr Ladies' Guild in Western House, Ayr.

27th (Thurs.). Annual Dance of the V.V.B.F. North of Scotland Division in the Northern Hotel, Aberdeen, 7.30 p.m.
Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, Essex, 7.30 p.m.

December

5th (Fri.). General Meeting of the Lincolnshire and District Division at the Grand Hotel, Peterborough, 2.15 p.m.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN LONDON

Wednesday, November 19th—No. 7, Mansfield Street

10.15 a.m. Finance Sub-committee.
11.45 a.m. Parliamentary and Public Relations Committee.
2.00 p.m. Small-Animals Committee.
4.00 p.m. Farm Livestock Committee.

Thursday, November 20th—No. 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Home Appointments Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, November 21st—Connaught Rooms

10.15 a.m. Council Meeting.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The R.C.V.S. and the B.V.A.

Sir,—It was with great pleasure that I noticed my good friend and ex-colleague, Raymond Mitchell, bursting into print (" Tribute from Canada "). However, I feel that I cannot let his unqualified praise of the R.C.V.S. and B.V.A. go unchallenged. His generous remarks must have been prompted by homesickness for the " fairy glens of South Yorkshire " or by a genial benevolence following his much improved status as a veterinary surgeon in North America!

It is on this last point that I cross swords with him in respect of the lavish praise for the two bodies concerned.

For instance, how do these organisations compare with the B.M.A.? Very poorly, I suggest. I cannot recall any past evidence of either the R.C.V.S. or B.V.A. contesting *strongly* for better working conditions or social advancement for the majority of their members, whether they be assistants in practice, teachers, or government employees. Most of us pay our yearly tribute of eight guineas and, apart from receiving that admirable journal, THE VETERINARY RECORD, and perhaps a chance to attend a congress once a decade, very little benefit is apparent.

Surely, if any bodies should carry responsibility for seeing that their members get a fair deal and a status comparable with veterinary surgeons overseas, it is the B.V.A. and R.C.V.S.? By this, I mean really getting to grips with employing bodies such as universities, government departments, and even principals of practices.

I know that some older members of the profession may think this savours too much of trade unionism. For myself, I can see nothing wrong in the principles of trade unionism being applied to professional bodies. Without, our profession will never achieve its aspirations of status and the decent living standards that go with it. If one condemns such principles, then the B.M.A. are already condemned.

From the government employees' point of view, it seems fantastic that we must needs join a block civil service union to press for better conditions of service. The onus is surely on our two veterinary organisations—even if it means increasing fees? Whether or not the B.V.A. or R.C.V.S. were consulted in the last major re-organisation of the government veterinary service, I do not know. If they were, then they must certainly possess a very small voice or a complete lack of interest—because as a result of the re-organisation veterinary surgeons have been further reduced in status.

In conclusion, may I add that I am not unaware of much good work done by the various committees of the B.V.A. and R.C.V.S. The spirit, I am sure, is willing, but the flesh is weak. The " flesh," in this instance, comprises the members of the profession, and the weakness is due to complete apathy.

For our veterinary organisations to receive praise from expatriates may be very pleasant; but both the B.V.A. and the R.C.V.S. should consider, for a moment, the conditions of service in this country which have caused many fellow members to emigrate.

Yours faithfully,
DONALD BASINGER.

" Dutch Cottage,"
Cold Norton,
Nr. Chelmsford,
Essex.
October 13th, 1958.

Brucella Infection in Man

Sir,—I am writing a book on Brucella Infection in Man and I am very anxious to reproduce in it a coloured photograph of a Brucella arm rash. May I, therefore, appeal through your columns to any veterinary surgeon who may suffer from such a rash, within the next four months, to inform me immediately so that I may visit him and take a coloured photograph of his arm.

I should be most grateful for help of this kind from my veterinary friends, who have always been so helpful to me in the past.

Yours faithfully,
WELDON DALRYMPLE-CHAMPNEYS.

3, Bryanston Square,
London, W.1.
Telephone : Ambassador 5197.
November 4th, 1958

A Suggested New Feature

Sir,—I wonder if the following suggestion commends itself to you?

It seems that many useful ideas and observations are lost to the profession because they are not fully supported by scientific facts. In consequence their authors are shy of publishing them because they are not " scientific." But it may be that this type of information would be of value in so far as it might lead to fruitful research or fill a missing gap in an existing piece of research.

If publication were allowed it might be best to insist that the information be presented in near-abstract form, and if the contributor wished, as well he might, he should be allowed to write anonymously. Surely, to a large extent, such information is the precursor of fruitful work from which the profession grows?

Yours faithfully,
EDWARD F. PECK.

The F.A.O. Mission,
Box 1039,
Addis Ababa,
Ethiopia.
October 23rd, 1958

A "Challenge"

Sir,—At a recent meeting of the Central Veterinary Society, Mr. C. E. Woodrow believed that: "The nation-wide use of egg-adapted canine distemper virus vaccines appeared to be setting up a barrier to the virus which might eventually eradicate the disease. There were, however, clinical indications that a mutation virus—against which vaccination offered only partial protection—might cause an epidemic of the disease in November or December this year."

The Metropolitan Region of the British Small Animal Veterinary Association has issued a "challenge" to Mr. Woodrow, that Association's President, to explain and support his statement.

Mr. Woodrow has accepted, and is picking up the glove over coffee at a dinner-meeting to be held at the Royal Society of Medicine, 1 Wimpole Street, on Wednesday, November 19th. Informality will be the key-note of this meeting and a cordial invitation is extended to everyone interested, including ladies, to come and dine and discuss. In your Notices column, Sir, are published the details of time, etc.

Yours faithfully,
OLIVER GRAHAM-JONES.

Zoological Society of London,
Regents Park.

November 1st, 1958.

Following the Articles of Association

Sir,—In reply to the letter from Mr. A. J. Beeson, dated October 22nd, which you published under the title above, may I explain the circumstances which led to the decision to defer, until Council had given further consideration, the venue of the 1960 Congress? At the Council meeting convened and held on the evening previous to the Annual General Meeting grave doubts were expressed by the members from the West of Scotland of the wisdom of holding the 1960 Congress in Glasgow owing to the lack of hotel and other accommodation. This warning of the difficulties to be faced in Glasgow, and the little time left to explore the possibilities of other venues, had the effect of producing only a very lukewarm recommendation for Glasgow to the Annual General Meeting. This, no doubt, prompted Mr. Beynon at that meeting (September 22nd), to propose that further thought be given to the question, and a decision deferred until further enquiries were made regarding accommodation and other facilities in Glasgow.

Mr. Baillie Russell, of Glasgow, then pointed out that although the delegates would be very welcome in that city hotel accommodation was very limited and would prove difficult, thus confirming the view of the West of Scotland representatives in Council.

Mr. A. J. Wright then suggested that the location of the 1960 Congress be reconsidered by Council, and that the Annual General Meeting delegate its authority to Council to decide the venue of the 1960 Congress. Mr. Beynon agreed with this alteration to his proposition, which was then put to the meeting and carried.

I hope, Sir, that this explanation will satisfy Mr. Beeson that the spirit of democracy, the constitution, and the well-being of our Association were not being abrogated—even in a light-hearted manner—and that due consideration was given to the feelings and the wishes of every member present.

Yours faithfully,
J. M. INGRAM.

Three Ways,
Limpsfield,
Oxted, Surrey.

November 3rd, 1958

"Tranquillising" for Shows

Sir,—In your last edition Mr. Caspari states that he is at a loss to understand my point of view. May I beg the courtesy of your columns again, in order to explain?

Surely Mr. Caspari will agree that temperament can be inherited, and also that a highly-strung, nervous temperament is undesirable. It follows that a dog whose temperament will not stand up to the stress of the show ring without tranquillising may pass on this undesirable temperament to his progeny. Therefore it is wrong to allow such dogs to win prizes, which they could not do without the aid of drugs, and in consequence of their success in the show-ring to be extensively bred from.

In my opinion faulty temperament, that is any nervousness which makes a dog unable to face stress, whether in the show ring or in traffic or merely as a family pet, as is the case with many dogs one meets to-day, is as much a fault as wrong tail carriage, or bad eye colour. I consider that the masking of temperamental defects in the show-ring by the use of drugs is on a par with the masking of faulty tail carriage by surgical interference, and I believe the Kennel Club share this opinion.

No veterinary surgeon should agree to assist in faking by means of his surgical skill, neither should he use his power to supply or prescribe drugs for a similar purpose.

Yours faithfully,
LUCY R. ROBINSON.

The Grey House,
Purleigh,
Nr. Chelmsford, Essex.

November 3rd, 1958.